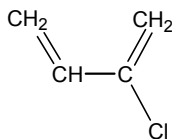


CHLOROPRENE
CAS No. 126-99-8

First Listed in the *Ninth Report on Carcinogens*



CARCINOGENICITY

Chloroprene is *reasonably anticipated to be a human carcinogen* based on evidence of benign and malignant tumor formation at multiple tissue sites in multiple species of experimental animals (NTP 467, 1998). Inhalation exposure of rats to chloroprene vapors induced increased incidences of neoplasms of the oral cavity, thyroid gland, and kidney in males and females, neoplasms of the lung in males, and neoplasms of the mammary gland in females. Inhalation exposure of mice to chloroprene vapors induced increased incidences of neoplasms of the lung, circulatory system (hemangiomas and hemangiosarcomas), forestomach, and harderian gland in males and females, kidney neoplasms in males, and neoplasms of the mammary gland, liver, Zymbal's gland, skin, and mesentery in females.

There is limited evidence for the carcinogenicity of chloroprene in humans. Data from two studies suggest that occupational exposure to chloroprene may increase cancer risk for digestive and lymphatic/hematopoietic tumors (Pell, 1978) and for liver, lung, and lymphatic tumors (Li et al., 1989).

ADDITIONAL INFORMATION RELEVANT TO CARCINOGENESIS OR POSSIBLE MECHANISMS OF CARCINOGENESIS

Recent studies on the genotoxicity of chloroprene have been uniformly negative; conflicting results with earlier studies have been attributed to differences in the age and purity of the test samples. Positive mutagenicity results of chloroprene in bacteria (Bartsch et al., 1975, 1979) were considered to be due to cyclic dimers that accumulate in aged samples (Westphal et al., 1994). At the exposure concentrations used in the cancer inhalation studies, chloroprene did not induce sister chromatid exchanges or chromosomal aberrations in mouse bone marrow cells nor did it increase the frequency of micronucleated erythrocytes in peripheral blood (Tice et al., 1988). Oxidation of chloroprene to epoxide intermediates has been postulated to occur based on the detection of alkylated derivatives of the trapping agent 4-(4-nitrobenzyl)pyridine in incubations of chloroprene and mouse liver microsomes (Bartsch et al., 1979). Chloroprene-induced lung and harderian gland neoplasms in mice had a high frequency of unique *K-ras* mutations (NTP 467, 1998). Chloroprene (chemical name: 2-chloro-1,3-butadiene) induced all of the types of tumors that were induced by 1,3-butadiene in mice except for lymphomas and ovarian neoplasms.

No data are available that would suggest that the mechanisms thought to account for tumor induction by chloroprene in experimental animals would not also operate in humans.

PROPERTIES

Chloroprene is a colorless liquid with a pungent, ethereal odor. It is slightly soluble in water and miscible in ethanol and diethyl ether (Lewis, 1992; HSDB, 1997). Chloroprene has a melting point of -130 °C and a boiling point of 59.4 °C (Johnson, 1979a; Lewis, 1992). It is a highly dangerous fire hazard. When exposed to heat or flame, it is explosive in vapor form (Lynch, 1997 personal communication). Chloroprene will rapidly auto-oxidize in air to form acidic materials and unstable peroxides, which catalyze exothermic polymerization of the monomer. It will also polymerize at room temperature to produce cyclic dimers or open-chain, high-molecular-weight products. When heated to decomposition, chloroprene emits hydrogen chloride (HSDB, 1997).

USE

Chloroprene is primarily used as a monomer in the production of the elastomer polychloroprene (neoprene), a synthetic rubber used in the production of automotive and mechanical rubber goods, adhesives, caulks, flame-resistant cushioning, construction goods, fabric coatings, sealants for dams or locks in waterways, roof coatings, fiber binding, footwear, and other applications requiring chemical, oil, or weather resistance, or high gum strength (IARC V.19, 1979; Johnson, 1979a, 1979b; Budavari, 1996; NTP 467, 1998).

PRODUCTION

In 1995, one producer of chloroprene for sale and distribution existed in the United States (SRIa, 1996). Other plants produced chloroprene for on-site use and processing, as a by-product of vinyl chloride production, or as a manufacturing impurity (TRI95, 1997). Used almost exclusively to produce polychloroprene, chloroprene is sold to only three U.S. companies for polychloroprene manufacture; less than 20 lb/yr is sold for research applications. The total estimated production of polychloroprene in 1986-1988 was approximately 250-300 million lb (113,000 to 136,000 Mg [metric tons]), and the volume in 1995-1996 was approximately 200-250 million lb (90,700 to 113,000 Mg) (Lynch, 1997 personal communication). These production numbers are higher than those given for U.S. polychloroprene shipments, which represent only off-site transfers, based on information from the International Institute of Synthetic Rubber Producers (e.g., 10,000 Mg in 1995-1996) (Chem. Eng. News, 1997).

EXPOSURE

Chloroprene is not known to occur naturally in the environment (HSDB, 1997). The primary route of potential human exposure to chloroprene is inhalation. The effluent and emissions from facilities that use chloroprene to produce polychloroprene elastomers are the main sources of environmental releases of chloroprene. Of 14 facilities reporting atmospheric releases of chloroprene in 1995, 8 plants reported individual atmospheric releases from 2 to 481,871 lb (0.0009 to 218.6 Mg), for a total release of 983,888 lb (446.3 Mg). Three plants in Kentucky, Texas, and Louisiana, each reporting atmospheric releases of >100,000 lb, accounted for most of the reported chloroprene releases. One of the sites is the producer and the other two sites convert chloroprene to polychloroprene. One of the 14 facilities also reported a chloroprene release consisting of 60,000 lb (27.2 Mg) by injection in deep wells, while another facility released 5,104 lb (2.315 Mg) to land (TRI95, 1997).

Volatilization is the primary mechanism of removal of chloroprene from water; chemical hydrolysis, adsorption to suspended solids or sediments, or bioaccumulation in aquatic animals is not expected to occur. If released to soil, chloroprene should be susceptible to removal by rapid volatilization and transport by leaching into groundwater. In the atmosphere, the primary mechanism of chloroprene removal is reaction with photochemically generated hydroxyl radicals with smaller amounts removed by reaction with ozone. Formaldehyde, 1-chloroacrolein, glyoxal, chloroglyoxal, and chlorohydroxy acids or aldehydes are expected products of these reactions (HSDB, 1997). The Urban Air Toxics Monitoring Program (UATMP), developed in 1987 by EPA, collected 349 samples from 12 sites every 12 days for 24-hour periods from March 1990 through February 1991. Chloroprene was identified in 88 of 349 samples (25.2%). The range of concentrations was 0.01 to 1.78 ppbv for samples in which chloroprene was identified with a mean of 0.26 ppbv ($0.94 \mu\text{g}/\text{m}^3$). The mean concentration based on all samples was 0.06 ppbv, where zero was used for samples not containing chloroprene (McAllister et al., 1991).

The primary potential occupational exposure to chloroprene will be that of workers manufacturing the compound or polychloroprene (NTP 467, 1998). Infante (1977) reported that an estimated 2,500 to 3,000 workers were exposed to chloroprene during its manufacture and polymerization. The National Occupational Exposure Survey (NOES), conducted by NIOSH from 1980 through 1983, reported that an estimated 17,700 workers, including 650 females, were potentially exposed to chloroprene or polychloroprene (NIOSH, 1990). Many workers were employed in auto repair services; their inclusion is probably attributable to their use of polychloroprene in belts, hoses, gaskets, and adhesives. Since residual monomer is appreciable only in polychloroprene latex ($\leq 0.5\%$ monomer), their inclusion may overestimate the actual number of workers exposed to chloroprene. DuPont Dow Elastomers estimated that fewer than 500 workers are exposed to chloroprene during manufacture. The company currently meets its internal control limit of 10 ppm, with the majority ($>95\%$) of the time-weighted exposures <2 ppm (Lynch, 1997 personal communication). ACGIH recommends a threshold limit value of 10 ppm ($36 \text{ mg}/\text{m}^3$) for chloroprene in the work environment (ACGIH, 1996).

REGULATIONS

Chloroprene is regulated by EPA under the Clean Air Act (CAA), Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), and Superfund Amendments and Reauthorization Act (SARA Title III, Section 313). The FDA allows the use of chloroprene in adhesives, assuming that there is a functional barrier between the adhesive and food or the exposure is the minimum acceptable under good manufacturing practice. Chloroprene polymers may be safely used in rubber products intended for repeated use in producing, manufacturing, packing, processing, preparing, treating, packaging, transporting, or holding food. NIOSH recommends a 15-minute ceiling value of 1 ppm ($3.6 \text{ mg}/\text{m}^3$) for chloroprene (Ludwig, 1994). OSHA has established a permissible exposure limit (PEL) of 25 ppm ($90 \text{ mg}/\text{m}^3$) as an 8-hour time-weighted average (TWA) for employee exposure to chloroprene. Regulations are summarized in Volume II, Table B-25.